Correspondence

The Editorial Board will be pleased to receive and consider for publication correspondence containing information of interest to physicians or commenting on issues of the day. Letters ordinarily should not exceed 600 words, and must be typewritten, double-spaced and submitted in duplicate (the original typescript and one copy). Authors will be given an opportunity to review any substantial editing or abridgement before publication.

Management of Stroke

TO THE EDITOR: I am writing regarding the January Medical Staff Conference, "Controversies in the Medical Management of Stroke,"[1] by Michael E. Charness, MD.

Wings was an appealing and insightful play about the effects of aphasic stroke upon a vibrant woman, and Geschwind's excellent review[2] of this play and of the consequences of stroke was both instructive and insightful. Charness's lucid and timely description of what we know and do not know about the medical management of stroke struck me similarly. Yet both Geschwind and Charness have failed to emphasize what is, in my view, one of the most important issues in patients who have suffered a completed stroke—the diagnosis and correction of problems which *led* to the stroke in the first place.

Charness's emphasis upon cardiogenic emboli notwithstanding, the majority of patients who suffer embolic cerebral infarction do so because of atheromatous involvement of the carotid bifurcation. This is generally on the basis of embolization of platelet-fibrin thrombi from ulcerative plaques at this extracranial site. While cardiac problems—myocardial infarction, congestive heart failure, arrhythmias—remain the leading overall cause of death in patients with cerebrovascular disease, the most prominent cause of death in stroke victims for the first five years after their cerebrovascular accident is, in fact, recurrent stroke.[3] Stroke victims' risk of recurrent stroke may be as high as 50% in the subsequent five years.[3,4] While these patients are being anticoagulated to diminish their chances of immediate recurrent stroke, it seems prudent to pursue an aggressive diagnostic approach, including contrast arteriography to delineate the presence of an extracranial lesion. A high-grade stenosis at the carotid bifurcation might be found, for example, or a complex ulcerative plaque—surgical relief of which might reduce *that* risk factor as a cause of recurrent stroke.

To be fair, stroke victims undergoing subsequent carotid operation are at higher risk for perioperative complications, and at least one analysis[5] has suggested that this increased risk of perioperative stroke and death essentially negates any benefit to be obtained by carotid endarterectomy. Other studies,[6] however, appear strongly to support the principle that expert operative treatment of extracranial carotid lesions in well-selected patients can be of substantial value in reducing the rate of recurrent stroke and death in stroke victims.

In short, to Charness's elegant discussion of the role of various pharmacologic manipulations in victims of acute stroke I would add a plea for early and aggressive diagnostic evaluation in search of operable extracranial vascular lesions in these high-risk patients.

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3. Robinson RW, Demirel M, LeBeau RJ: Natural history of cerebral thrombosis—9-19 year follow-up. J Chronic Dis 1968; 21:221-230

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Type II Diabetes Mellitus

TO THE EDITOR: After the review "Pathogenesis of Impaired Glucose Tolerance and Type II Diabetes Mellitus—Current Status"[1] was accepted for publication, the Mayo Clinic group published a report updating their experience in patients with impaired glucose tolerance and type II diabetes (which they do not separate). In their first paper,[2] in which they studied ten patients with a mean fasting plasma glucose concentration of 182 mg per dl, the following were found: (1) decreased insulin binding, (2) a shift of the dose-response curve to extracellular insulin concentrations to the right (decreased sensitivity), (3) normal maximal responses (normal responsiveness) and (4) a normal dose-response curve to the amount of bound insulin (normal coupling between insulin binding and the effect of the bound insulin). Hence, they concluded that the mechanism of insulin antagonism in these patients was due to a receptor defect.

In their second report,[3] they added seven patients to their series, the effect of which was to change their results and conclusions. The mean fasting plasma glucose concentration in these 17 patients was 178 mg per dl so that the degree of abnormal carbohydrate metabolism did not change by adding these new patients. However, when the results of all 17 patients were analyzed, the following were found: (1) decreased insulin binding (which the authors ascribed to down regulation), (2) decreased sensitivity, (3) decreased responsiveness

and (4) impaired coupling between insulin binding and action. Hence, they now conclude that the mechanism of insulin antagonism in these patients is primarily due to a postreceptor defect.

These more recent data shift the evidence even more strongly to favor the view that a postreceptor defect characterizes patients with even mild type II diabetes mellitus.

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Cinnamon Oil Burn

TO THE EDITOR: Recently an 11-year-old boy, a sixth grade student of apparently normal intelligence, presented at the University of New Mexico pediatrics clinic with a 10 by 12 cm second degree burn on his posterior thigh. This blistered area was surrounded by a 3 to 4 cm first degree burn. The injury was the result of a cinnamon oil spill from a broken vial in his rear pants pocket. The area had remained unwashed for 48 hours, and smelled strongly of cinnamon.

In reviewing the literature and contacting Poisindex Information System, I was able to find no previous report of such an incident. It has long been known that cinnamon oil contains many irritants, cinnamaldehyde being the most strongly implicated. The oil is known to irritate mucous membranes, skin and gastrointestinal tract, as well as cause hypersensitivity reactions.

The fact that it can also burn under certain circumstances may be of importance in the Southwest. It is a common practice among school children in this part of America to soak toothpicks in cinnamon oil obtainable from drugstores and supermarkets. The toothpicks are then chewed for the flavor.

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Protective Helmets for Infants and Children on Bicycles

TO THE EDITOR: The correspondence from Drs James, Buchta and Stein[1] in the March issue draws proper conclusions from improper data. They point out that 78% of bicycle fatalities occur in children but fail to indicate what percentage of bicycle riders are children.

Their plea is for the use of specially designed helmets that protect infants and toddlers (one must presume the child to be riding behind a parent in an infant seat). However, they gave no statistics concerning the scope of the problem in this population or even if there is a problem in this population.

The statements presented in the last paragraph (excluding the last sentence) seem completely unrelated to the subject suggested by the title.

I laud efforts by those who encourage bicyclists (of all ages) to wear protective head gear and congratulate Bell Helmet, Inc, Norwalk, Calif, for designing and producing helmets for infants and small children, but I think that statistics presented to support a claim in medical literature should be related to that claim.

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REFERENCE

1. James HE, Buchta R, Stein M: A protective helmet for infants and children who accompany their parents on bicycles (Correspondence). West J Med 1985 Mar; 142:403

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Dr James Replies

TO THE EDITOR: We take note of the correspondence of Dr Michael T. Long in reference to the conclusions of the information on protective helmets for infants and children. Dr Long correctly points out that we did not break down the percentage of bicycle riders that are children. It would be very advantageous to have these data, but they are not available to my knowledge. I also wholeheartedly agree that it would be more appropriate to include statistics on the subgroups of the various populations and the types of injuries suffered. Unfortunately, once more, the data are not available.

We do know that the most common cause of hospital admission and the largest expense of health care in pediatrics is trauma. The thrust of our presentation was to make the primary care physicians caring for children (and the families who request the care) aware that there is a protective device for a child's cranium that was not previously available.

I wish to laud the efforts of two concerned pediatricians, Dr Buchta and Dr Stein, in cooperating with us and with the manufacturer in trying to make the general population and physicians aware that this is one preventive element within their reach. As always, in trauma, prevention should be our major objective.

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The True Role of Physicians

TO THE EDITOR: I wish to commend you for your editorial comments in both the February and March issues.[1-3] Your remarks present, I believe, a perspective from which we can take a broader view of our obligations to our patients and to society. Further and properly, I submit, your remarks aim beyond our one-on-one physician-patient relationships and have broad bioethical implications that potentially could not only dispel the "stereotype" of organized medicine but also contribute to efforts toward a safer, peaceful planet. This will not be an easy task, for despite the AMA survey that you reported, my own experience over the years convinces me that the self-serving image of professional associations is a widely perceived one. However, I feel very strongly that the effort will be worthwhile, and that at this particular moment it is much needed. As you so correctly point out, "real power" does not lie with organized medicine, but with society. Despite pressures from government, the media and special interests, if we fulfull our obligations to provide high quality care that is accessible to all, and affordable, then society, the